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Evaluation of the relationship between the RDW/albumin ratio and coronary artery ectasia

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ABSTRACT

Aims: Coronary artery ectasia (CAE) is a vascular abnormality associated with inflammation and adverse cardiovascular outcomes. The red blood cell distribution width-to-albumin ratio (RAR) has emerged as a novel biomarker reflecting systemic inflammation and nutritional status. This study aimed to evaluate the relationship between RAR and the presence of CAE.

Methods: In this retrospective study, 80 patients diagnosed with CAE via coronary angiography and 80 age-and sex-matched healthy controls were included. Laboratory parameters, including RDW, serum albumin, and RAR, were analyzed. The Mann-Whitney U test was used for group comparisons, and significance was set at $p<0.05$. Patients with active infection, severe organ failure, or malignancy were excluded.

Results: Demographic and clinical variables were similar between groups ($p>0.05$). Median (IQR) serum albumin levels were significantly lower in the CAE group compared to controls [41.0 (39.0–43.9) g/L vs. 43.9 (42.0–45.7) g/L; $p<0.001$]. Although RDW alone did not differ significantly between groups ($p=0.448$), the RAR was significantly higher in CAE patients [0.375 (0.338–0.410)] than in controls [0.355 (0.330–0.390)] ($p=0.036$). Additional findings included reduced lymphocyte and platelet counts and elevated monocyte counts and mean platelet volume (MPV) in the patient group, indicating a pro-inflammatory state.

Conclusion: The RDW/albumin ratio was significantly elevated in patients with CAE, suggesting that it may serve as a simple, inexpensive, and effective inflammatory biomarker in this population. While RDW and albumin are routinely available in clinical practice, their combined interpretation via RAR may provide additional insight into the inflammatory status of patients with CAE. Further prospective, large-scale studies are warranted to confirm these findings and clarify the prognostic value of RAR in CAE.

Keywords: Coronary artery ectasia, RDW/albumin ratio, inflammation, red blood cell

INTRODUCTION

Coronary artery ectasia (CAE) is a vascular anomaly characterized by segmental or diffuse dilation of a coronary artery, with the affected segment exhibiting a diameter at least 1.5 times greater than that of an adjacent normal coronary segment.[1] The prevalence of CAE varies between 0.3% and 5% across different populations.[2] Although often identified incidentally, its clinical relevance has gained attention due to its potential association with adverse cardiovascular outcomes.[3]

The most common method of diagnosis is invasive coronary angiography. Quantitative measurements improve the evaluation's impartiality and make it possible to determine luminal dilatation more precisely, especially when it comes to vascular diameter increase. Ectasia is categorized as little (less than 5 mm), moderate (5 to 8 mm), and large/"giant" (more than 8 mm) in the literature.[1]

The pathogenesis of CAE is multifactorial. While atherosclerosis remains the most common underlying cause accounting for more than half of all cases other etiologies, including congenital anomalies, vasculitis, connective tissue disorders, and infections, have also been reported.[1-4] Recent evidence underscores the role of inflammatory and immune-mediated processes, as demonstrated by elevated levels of matrix metalloproteinases (particularly MMP-9), tumor necrosis factor-alpha (TNF-α), and interleukins such as IL-1β and IL-6. These mediators contribute to extracellular matrix degradation and vascular remodeling. Additionally, lipid profile disturbances such as elevated low-density lipoprotein cholesterol (LDL-C) and reduced high-density lipoprotein cholesterol (HDL-C) have been implicated in the pathophysiology of CAE.[5]

Clinically, CAE presents across a wide spectrum—from asymptomatic cases to those manifesting with stable angina,

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acute coronary syndromes (ACS), or myocardial infarction. These manifestations are believed to result from disturbed coronary hemodynamics, leading to turbulent blood flow, endothelial dysfunction, and thrombus formation within the dilated segments.[1-5] The diagnosis is primarily established via coronary angiography, which remains the gold standard for assessing the morphology and extent of ectasia.[1] Nevertheless, non-invasive modalities such as coronary computed tomography angiography (CCTA) and magnetic resonance angiography (MRA) have gained prominence, offering high-resolution, three-dimensional imaging of the coronary anatomy, supporting both diagnosis and longitudinal monitoring.[4] Importantly, patients with CAE face an increased risk of distal embolization and recurrent myocardial infarction, even in the absence of significant obstructive coronary artery disease.[6]

Management of CAE remains challenging due to the lack of universally accepted treatment guidelines. Therapeutic approaches are often tailored to the individual, focusing on the use of antiplatelet agents, anticoagulation in selected cases, and rigorous control of modifiable cardiovascular risk factors. Invasive interventions, such as percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG), are typically reserved for patients with significant stenosis or ischemia-inducing lesions.[7]

In conclusion, CAE represents a complex and frequently underrecognized clinical entity with diverse implications. Given its heterogeneous etiology, variable clinical presentation, and inconsistent therapeutic response, further research is necessary to elucidate its pathophysiology, refine diagnostic criteria, and establish evidence-based management strategies.

The utilization of biomarkers in the diagnosis and prognosis of cardiovascular diseases has significantly increased in recent years. Within this context, erythrocyte distribution width (RDW) and serum albumin levels have emerged as important parameters that reflect systemic inflammation and overall physiological status. RDW indicates the degree of variation in erythrocyte volume, and its elevation has been associated with inflammation, oxidative stress, and malnutrition.[8] Conversely, reduced serum albumin levels have been linked to chronic inflammation, malnutrition, and cardiovascular events.[9]

More recently, the RDW/albumin ratio (RAR) has been proposed as a novel biomarker that reflects the combined effect of these two parameters. RAR is believed to hold prognostic value, particularly in conditions such as stroke. Moreover, RAR has been associated with increased morbidity and mortality across various cardiovascular diseases.[10]

However, the association between RAR and more specific cardiovascular conditions such as CAE has not been thoroughly explored. Accordingly, investigating the clinical significance and potential prognostic value of RAR in CAE patients represents an important area of study. Recent research suggests that hematological parameters may contribute to the pathophysiology of CAE. Therefore, this study aimed to assess the relationship between the RDW/Albumin ratio and the presence of CAE, with the hypothesis that inflammation may play a contributing role.

METHODS

The study was conducted with the permission of Bolu İzzet Baysal University Non-interventional Clinical Researches Ethics Committee (Date: 06.05.2025, Decision No: 2025/208). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

In this retrospective study, laboratory blood results recorded in the hospital automation system were analyzed for patients diagnosed with CAE who underwent coronary angiography at İzzet Baysal Training and Research Hospital, along with data from a healthy control group. Blood RDW, albumin levels, RDW/Albumin ratio, and several additional hematological parameters were compared between CAE patients and controls. Data were evaluated from 80 patients with CAE and 80 control individuals. Blood samples were processed using an automated hematology analyzer. Group comparisons were conducted using the Mann-Whitney U test ($p<0.05$), and correlation analyses were performed to assess associations with CAE severity.

Exclusion criteria included patients with severe valvular disease, advanced heart failure (EF<30%), severe acute or chronic liver and/or kidney failure, recent coronary intervention within the past 3 months, advanced chronic lung disease, age below 18 years, pregnancy, history of thrombolytic therapy, severe inflammation, hematological or oncological malignancies, and severe anemia.

Statistical Analysis

The data analysis was conducted using SPSS software (SPSS 22.0 for Windows, IBM Corp., Chicago, IL, USA). The Kolmogorov-Smirnov test was used to evaluate normality of distribution. For normally distributed variables, comparisons were made using the t-test and results were expressed as mean±standard deviation. For variables with non-normal distribution, the Mann-Whitney U test was used, and results were presented as median and interquartile range (IQR, 1st-3rd quartile values). Categorical variables were compared using the Chi-square test.

RESULTS

A total of 160 participants were included in the study, with 80 individuals in the patient group and 80 in the control group. **Table 1** outlines the baseline demographic and clinical characteristics of the study population. The median (IQR) age was higher in the patient group [63 (51.75–69) years] than in the control group [59 (52.75–66) years]; however, this difference was not statistically significant ($p = 0.097$). Sex distribution was identical between groups, with 40 females (50%) and 40 males (50%) in each group, indicating no sex-based imbalance ($p>0.999$).

With regard to lifestyle factors and comorbidities, smoking prevalence was nearly the same between groups (28.75% in patients vs. 27.5% in controls), with no statistically significant difference ($p=0.860$). Similarly, diabetes mellitus was present

Table 1. Demographic and clinical characteristics of patients and controls

		Control	Patient	p value
Age, years		59 (52.75-66)	63 (51.75-69)	0.097
Sex	Female	40 (50%)	40 (50%)	>0.999
	Male	40 (50%)	40 (50%)	
Smoking	Absent	58 (72.5%)	57 (71.25%)	0.860
	Present	22 (27.5%)	23 (28.75%)	
Diabetes mellitus	Absent	53 (66.25%)	52 (65.0%)	0.868
	Present	27 (33.75%)	28 (35.0%)	
Hypertension	Absent	40 (50%)	32 (40%)	0.204
	Present	40 (50%)	48 (60%)	

Descriptive statistics were presented as median (1st–3rd quartile) for continuous variables or number (percentage) for categorical variables. Comparisons were performed using the Mann–Whitney U test or the Pearson Chi-square test, as appropriate

in 35.0% of patients and 33.75% of controls (p=0.868). The prevalence of hypertension was slightly higher in the patient group (60%) than in the control group (50%), though this difference was not statistically significant (p=0.204). These findings indicate that both groups were well-matched demographically and clinically, thereby reducing the risk of confounding (Table 1).

Laboratory parameters are presented in Table 2. Several hematological indices demonstrated statistically significant differences. Lymphocyte counts were significantly lower in the patient group [2.03 (1.58–2.50)×10^9/L] compared to the control group [2.50 (1.92–3.01)×10^9/L] (p<0.001). Monocyte levels were significantly elevated in patients [0.55 (0.42–0.70)×10^9/L] relative to controls [0.47 (0.36–0.57)×10^9/L] (p=0.004). Neutrophil counts did not significantly differ between the two groups [4.06 (3.44–5.16) vs. 3.96 (3.42–4.93)×10^9/L, p=0.416].

Hemoglobin (HGB) levels were significantly lower in patients (13.5±5.5 g/dl) than in controls (14.0±5.1 g/dl) (p=0.040). Platelet (PLT) counts were also significantly reduced in the patient group [230.5 (194.8–267.5)×10^9/L] compared to controls [253.5 (212.8–298.8)×10^9/L] (p=0.028). Mean platelet volume (MPV) was significantly elevated in patients [8.45 (7.55–9.45) fL] versus controls [7.93 (7.52–8.67) fL] (p=0.029). Hematocrit (HCT) values showed a slight, non-significant decrease in patients [41.2 (37.65–45.08)%] relative to controls [42.25 (40.00–45.2)%] (p=0.071).

Serum albumin levels were significantly lower in the patient group [41.0 (39.0–43.9) g/L] compared to the control group [43.9 (42.0–45.7) g/L] (p<0.001). RDW did not significantly differ between groups [15.3 (14.40–16.43)% in patients vs. 15.45 (14.8–16.3)% in controls, p=0.448]. However, the RDW/albumin ratio was significantly higher in patients [0.375 (0.338–0.410)] than in controls [0.355 (0.330–0.390)] (p=0.036), suggesting that this ratio may serve as a more sensitive marker than RDW alone (Table 2).

To further illustrate the distribution of key variables, violin plots were generated (Figure). These plots depict the distributions of albumin, RDW, and RDW/albumin ratio across both groups. Figure 1a shows a clear leftward shift in albumin values in the patient group, indicating lower levels. Figure 1b displays RDW distributions, which appear similar between groups, consistent with the non-significant p-value. Figure 1c highlights a higher RDW/albumin ratio in patients, confirming the statistical findings. The violin plots, through kernel density estimation, visually emphasize central tendency and variability, supporting the numerical results.

DISCUSSION

This study investigated the association between RAR and the presence of CAE. Our findings demonstrate that elevated RAR levels are significantly associated with CAE, suggesting that RAR may function as a novel inflammatory biomarker in this clinical context.

The pathophysiology of CAE is multifaceted and not yet fully elucidated; however, inflammation is recognized as a critical contributor to its development. Previous studies have shown that patients with CAE exhibit higher levels of inflammatory markers, such as C-reactive protein (CRP) and interleukin-6 (IL-6), compared to individuals with normal coronary arteries.[11] RDW, which reflects the variability in red blood cell size, has been identified as an independent predictor of various cardiovascular diseases and is thought to mirror underlying inflammatory activity.[8] Similarly, hypoalbuminemia has been associated with poor nutritional status and systemic

Table 2. Laboratory parameters of patients and controls

	Control	Patient	p value
White blood cell (WBC), ×10^9/L	7.23 (6.25-8.68)	7.33 (6.01-8.30)	0.700
Lymphocyte, ×10^9/L	2.50 (1.92-3.01)	2.03 (1.58-2.50)	<0.001
Monocyte, ×10^9/L	0.47 (0.36-0.57)	0.55 (0.42-0.70)	0.004
Neutrophil, ×10^9/L	3.96 (3.42-4.93)	4.06 (3.44-5.16)	0.416
Hematocrit (HCT), %	42.25 (40.00-45.2)	41.2 (37.65-45.08)	0.071
Hemoglobin (HGB), g/dL	14.0±5.1	13.5±5.5	0.040
Platelet count (PLT), ×10^9/L	253.5 (212.8-298.8)	230.5 (194.8-267.5)	0.028
Mean platelet volume (MPV), fL	7.93 (7.52-8.67)	8.45 (7.55-9.45)	0.029
Red cell distribution width (RDW), %	15.45 (14.8-16.3)	15.3 (14.40-16.43)	0.448
Albumin, g/L	43.9 (42.0-45.7)	41.0 (39.0-43.9)	<0.001
RDW/albumin ratio	0.355 (0.330-0.390)	0.375 (0.338-0.410)	0.036

Descriptive statistics were presented as mean±standard deviation or median (1st–3rd quartile), and comparisons were made using Student's t-test or the Mann–Whitney U test, as appropriate

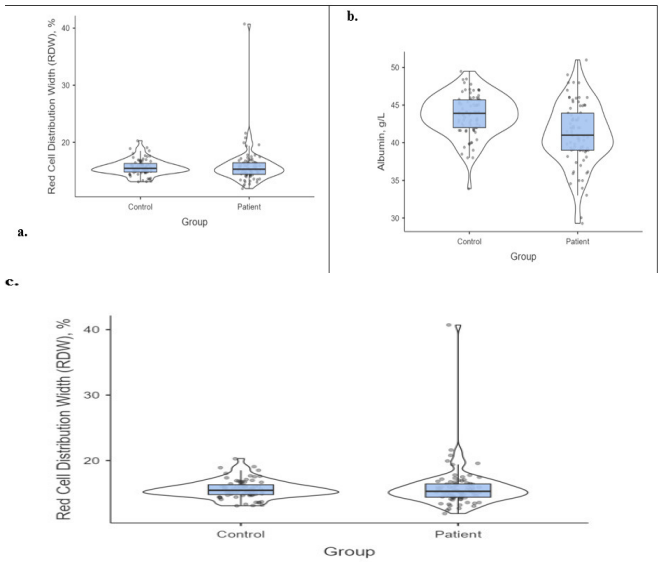

Figure. Violin plots comparing albumin (**a**), red cell distribution width (RDW) (**b**), and RDW/albumin ratio (**c**) between control and patient groups. In each plot, the central box represents the interquartile range (25^{th}–75^{th} percentile), the horizontal line within the box indicates the median, and the thin vertical lines ("whiskers") extend to 1.5 times the interquartile range. Individual data points are shown as dots, and the width of the violin reflects the kernel density estimation, indicating the distribution of values

inflammation, both of which correlate with unfavorable cardiovascular outcomes.[12]

Emerging research highlights the involvement of systemic inflammation and oxidative stress in the vascular remodeling observed in CAE, as demonstrated by elevated levels of CRP, IL-6, and TNF-α.[13] Given these associations, composite biomarkers such as the RDW/albumin ratio (RAR) may also hold relevance in this disease process. This retrospective cohort study evaluated the prognostic utility of RAR in critically ill patients diagnosed with both coronary heart disease (CHD) and diabetes mellitus (DM). The results demonstrated that elevated RAR values were significantly associated with increased in-hospital mortality. These findings underscore the potential of composite indices, such as RAR, to capture multidimensional physiological disturbances and enhance risk stratification in complex cardiovascular conditions.[14]

The integration of RDW and albumin into a single ratio the RAR provides a more holistic assessment of a patient's inflammatory and nutritional state. Recent studies have highlighted the prognostic value of RAR in several cardiovascular conditions, including acute myocardial infarction and heart failure.[15] However, to the best of our knowledge, this is the first study to examine the relationship between RAR and CAE.

Our findings align with the results of Li et al.,[16] who reported that elevated RDW levels are associated with the presence of CAE. Additionally, the inverse relationship between serum albumin and inflammation has been well established in atherosclerotic diseases, further supporting the biological plausibility of our results.[9] Cai et al.[17] focused on developing a predictive model for MACE in patients with CAE and found that certain clinical and angiographic features are associated with an increased risk of adverse cardiovascular events. The authors emphasized the importance of early risk stratification in these patients.

In a comprehensive cohort study involving 12.765 participants, Liu et al.[18] identified a non-linear, positive association between elevated RAR levels and increased risks of cardiovascular disease (CVD), all-cause mortality, and cardiovascular mortality. Notably, glycated hemoglobin (HbA1c) partially mediated the relationship between RAR and CVD, suggesting a link between glycemic control and inflammatory status in cardiovascular outcomes.

Similarly, Li et al.[19] demonstrated that higher RAR values independently predicted one-year mortality in intensive care unit (ICU) patients with heart failure. The study also highlighted RAR's potential to enhance prognostic accuracy when integrated with established scoring systems like SOFA and APACHE II.

In a meta-analysis conducted in 2025, RAR was identified as a novel and independent prognostic marker that is significantly associated with increased all-cause mortality in patients with cardiovascular disease.[20]

The clinical implications of these findings are substantial. RAR is a simple, inexpensive, and routinely accessible laboratory parameter that can be derived from standard blood tests. Its potential utility as a biomarker for CAE could facilitate earlier diagnosis and improved risk stratification, thereby enhancing clinical decision-making and patient outcomes.

Limitations

Nonetheless, our study has certain limitations. The cross-sectional nature of the design restricts the ability to draw causal inferences between elevated RAR and the presence of CAE. Moreover, the relatively small sample size necessitates further large-scale, prospective studies to confirm our findings and to evaluate the clinical utility of RAR in routine practice. Detailed information on patients' history of prior coronary interventions was not consistently available, which might have had a minimal or indirect effect on the findings.

CONCLUSION

In this study, we analyzed hematological and biochemical parameters in patients with CAE, focusing on albumin, RDW, and particularly the RDW/albumin ratio. While RDW alone did not differ significantly between groups, the RDW/albumin ratio was notably higher in patients, indicating its potential as a more sensitive marker of systemic inflammation or subclinical disease. The CAE group also exhibited lower albumin, reduced lymphocyte and platelet counts, and higher monocyte levels and MPV, collectively reflecting an underlying inflammatory or cardiovascular risk profile. These findings support the use of integrated indices like the RDW/albumin ratio over isolated values in clinical assessment. Given its ease of access and routine availability, this ratio may enhance clinical insight into CAE-related inflammation, though larger prospective studies are needed to confirm its prognostic relevance.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was initiated with the approval of the Bolu Abant İzzet Baysal University Non-interventional Clinical Researches Ethics Committee (Date: 06.05.2025, Decision No: 2025/208).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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